STUDY OF RECOVERY PATTERN OF APHASIA IN STROKE PATIENTS

Dissertation submitted to THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY for

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Coimbatore

CERTIFICATE

This is to certify that the Dissertation entitled "STUDY OF RECOVERY PATTERN OF APHASIA IN STROKE PATIENTS " here with submitted by Dr. A. AKILA, Post graduate in General Medicine,Coimbatore Medical College to the Tamilnadu Dr.M.G.R Medical University is a record of a bonafide research work carried out by her under my guidance and supervision from July 2007 to June 2008.

Dr. Nedumaran, MD, DM
Professor & Unit chief,
Department of Medicine.

Dr. Umakanthan, MD
Professor & Head,
Department of Medicine.

DEAN,
Coimbatore Medical College,
Coimbatore.

DECLARATION

I solemnly declare that the Dissertation titled "STUDY OF RECOVERY PATTERN OF APHASIA IN STROKE PATIENTS", was done by me at Coimbatore Medical College and Hospital during the period from Jul '07 to Jun'08 under the guidance and supervision of Prof. Dr. Nedumaran, MD, DM.

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University towards the partial fulfillment of the requirement for the award of MD Degree (Branch I) in General Medicine.

Place : Coimbatore

Date : **Dr. A. Akila**

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The T.N. Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate	:	Dr. A. Arila
Course	:	M.D. (General Medicine)
Period of Study	:	2006 - 2009
College	:	Coimbatore Medical College
Dissertation Topic	:	Study Of Recovery Pattern Of Aphasia In Stroke Patients

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation is accepted / ~~Not accepted~~ and you are permitted / ~~Not Permitted~~ to proceed with the above Study.

Coimbatore - 14.

Date : 5.3.2008

Secretary
Ethics Committee

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CONTENTS

1. INTRODUCTION

Stroke is defined as a focal or at times global neurological impairment of sudden onset, lasting more than 24 hrs and of vascular origin[1]. Cerebro vascular accidents rank among the leading causes of death and physical disability in general population. Of the stroke population 20 – 30 % suffer from communication deficits and aphasia[2,3] due to brain tissue damage. This interferes adversely with the patient's physical, mental and occupational rehabilitation. So thorough knowledge of aphasia is essential for stroke rehabilitation.

Aphasia is defined as a disorder of language that is acquired secondary to brain damage (Alexander and Benson -1997)[4].

Many studies have shown favorable outcome in aphasic patients of post traumatic etiology[5] .Few aphasia recovery studies have been done with stroke patients also[6,7].

A preliminary study was done regarding the recovery of aphasia by Vijayaraghavan et al in 1989 on 16 cases. Based on that study, a modified protocol suiting the needs of CMC patients with the concurrence of specialists was designed and this study was conducted to analyse the recovery pattern of aphasia in acute stroke patients.

2. AIM OF THE STUDY

The objectives of this study are:

1. To study the time course of spontaneous recovery of aphasia in stroke patients.
2. To compare the recovery patterns between different types of aphasia .
3. To evaluate the factors influencing the recovery of aphasia.
4. To correlate the clinical syndrome of aphasia with the anatomical substrate in the CT scan.

3. REVIEW OF LITERATURE

3.1 DEFINITION

Language refers to perception of verbal sensory stimuli, the integration with prior knowledge and activation of verbal response mechanisms. Neural substrate of language is composed of a distributed network centered in the perisylvian region of left hemisphere. Damage to any one of these components or interconnections leads to language disturbances.

Speech is the neuro mechanical process of articulation. Speech disturbances are produced by central and peripheral mechanisms.

Aphasia is best defined as disorder of previously normal language abilities due to brain damage[8].

Aphasic patients can not convert the nonverbal mental representations of thought in to the symbols and grammatical organization of language[9].

Aphasia affects multiple aspects of language. These include naming, word choice, comprehension, repetition, spelling, syntax (grammatical structure of sentence), lexicon (collection of words with meaning) and the morphology of words[9]. Reading , writing and copying may also be involved.

People with aphasia may have problems speaking, understanding speech, reading and writing. These problems can range from mild to severe in nature.

Aphasia does not generally affect the ability to think, reason and understand .Most aphasics know what they want to say –they have trouble putting their thoughts into words.They are not able to understand non verbal forms of communication such as gestures and facial expressions.

Aphasia can take many forms. Some aphasics have word finding difficulties (anomia). Some respond to questions by repeating (echolalia). Others use invented words (neologisms), or get stuck on a certain word, repeating it over and over (perseveration). Paraphasic errors ,in which other words are substituted are also common.

External speech ,or exophasy, means the expression of thoughts by spoken or written words and the comprehension of the spoken or written words of others.

Inner speech or endophasy, is the silent process of thought and the formulation in our minds of unuttered words on which thought depends. So thought and language are inseparable.

Other abnormalities include aprosody (loss of emotional content of speech) which occurs with non-dominant hemisphere lesions.

3.2 HISTORICAL BACKGROUND

Paul Broca published a post mortem study on a patient with aphasia in 1861. The lesion was in the posterior portion of inferior frontal gyrus – Broca's area. Later Broca underlined the fact that the left hemisphere is dominant for language.

Carl Wernicke published "Der Aphasische Symptometon complex" in 1874[10] and brought new pathologic and clinical findings in aphasic patients.

Wernicke differentiated between fluent aphasias with abnormal language and Broca's with gross paucity of language and established existence of fluent aphasias with poor comprehension, it was due to lesion in posterior portion of superior temporal gyrus – Wernicke's area.

Wernicke's paper marked a turning point and the succeeding years stimulated careful search for distinguishable clinical syndromes and pathologic lesions.

3.3 TYPES OF APHASIA

Based on speech output, aphasia is divided into fluent and and non-fluent types. Non-fluent aphasics have decreased word output, altered rhythm, dysarthria and reduced phrase length. They speak few words with lot of effort and convey the content properly.

Fluent aphasics have normal or increased word output with normal rhythm and articulation. Phrase length is normal or increased. Paraphasias and neologisms appear. Even with good word output they convey little information.

Dysarthria is weakness or paralysis of muscles of face, mouth, neck and throat due to brain injury and speech will be slurred and unintelligible. It may occur with or without aphasia.

ALGORITHM FOR APPROACH TO APHASIA

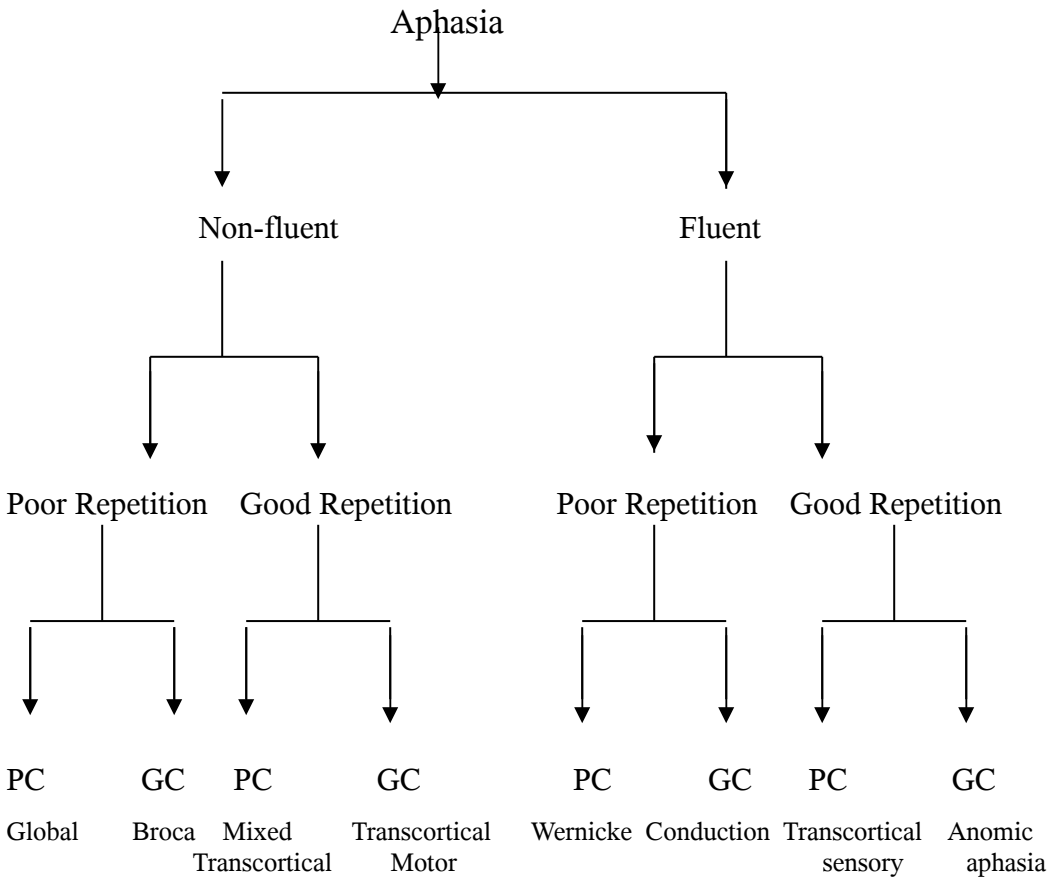


TABLE 1

DIFFERENTIAL DIAGNOSIS OF THE MAIN TYPES OF APHASIA

Types of Aphasia	Speech	Comprehension	Capacity for Repetition	Other Signs	Regions Affected
Broca's	Non-fluent effortful	Intact	Impaired	Right hemiparesis	Left frontal (lower posterior)
Wernicke's	Fluent	Impaired	Impaired	No motor signs	Left temporal (posterior and superior)
Conduction	Fluent	Intact	Impaired	Often none	Left supramarginal gyrus or left auditory cortex and insula
Global	Scant	Impaired	Impaired	Right hemiplegia	Left perisylvian lesion
Transcortical motor	Nonfluent explosive	Intact	Intact		Anterior or superior to Broca's area, may involve part of Broca's area
Transcortical sensory	Fluent scant	Impaired	Intact		Area surrounding Wernicke's area
Mixed transcortical	Non fluent echolalia	Impaired	Intact		Large, watershed infarction of left hemisphere or both hemispheres that spare the perisylvian cortex
Anomic	Fluent word finding pauses circumlocution	Intact	Intact	None	Left angular gyrus

3.3.1 Fluent aphasias:

a. Wernicke's aphasia

In Wernicke's aphasia, the word output is fluent and incomprehensible. Comprehension severely impaired. The output is voluminous but not informative, paraphasias, neologisms, circumlocution common. Repetition is disturbed equally. Naming is also impaired. Reading is always disturbed and the degree of disturbance parallels the disturbance of spoken language comprehension. Writing is always is abnormal. The patient usually has no paresis and writes with dominant hand. Wernicke's aphasia comprises about 15% of the aphasia population.

b. Conduction aphasia

Patients with conduction aphasia understand everything but have difficulty in uttering the right sounds. They repeat phrases or sentences poorly. Writing is usually impaired. Reading ability varies. Buccofacial apraxia is frequent. They may have a right homonymous hemianopia. The lesion is located in the arcuate fasciculus. Diagnosis is based on discrepancy between relatively preserved comprehension and impaired repetition. Conduction aphasia constitutes 10% of aphasic patients.

c. Anomic aphasia

This is the minimal dysfunction syndrome of language network, also called as amnestic aphasia. Articulation, Comprehension and Repetition are intact. The main disturbance consists of difficulty in naming on confrontation and in word finding in spontaneous speech and spelling is also impaired. Language output is fluent, but paraphasic, circumlocutious and uninformative. Comprehension of spoken language may vary from completely normal to impaired. Anomic aphasia represents 5% of the cases of aphasia.

d. Transcortical sensory aphasia

Here repetition is good but comprehension is impaired. There may be echolalia where patients repeat the words addressed to them. This syndrome is found in 2% of aphasic patients.

3.3.2 Non-fluent aphasias

a. Broca's aphasia

This is characterized by reduced output in both speech and writing. Comprehension appears intact on informal examination. In some cases comprehension is more severely impaired resulting in overlap between this form of aphasia and global

aphasia. Some patients initially present with global aphasia and improve in their ability to understand and evolve into Broca's aphasia. Repetition is always abnormal. Naming is abnormal. They have great difficulty in reading and writing. In addition to right hemiparesis, right facial weakness they have Buccofacial apraxia. Insight is preserved. They can sing normally and vision is also normal. About 20% of all patients with aphasia have Broca's aphasia.

b. Global aphasia

This produces the most severe deficit. They show severe impairment of all language functions. They understand few questions and commands and repeat only the simplest sounds. Reading, writing is impaired. Other signs include right hemiplegia, right hemisensory loss and homonymous hemianopia. Global aphasia is the most common type of aphasia (20-25%).

c. Transcortical motor aphasia

Patients with this syndrome have markedly reduced word output with relatively intact comprehension. Naming is quite good. Repetition is perfect. Reading is preserved and writing is nearly always impaired.

Disconnection (dissociative) syndromes

This term refers to certain disorders of language that results not from lesions of the cortical language areas themselves but from an apparent interruption of association pathways joining the primary receptive areas to the language areas[11].

Aphasia due to lesions that separate the receptive parts of the language mechanism from purely motor ones –conduction aphasia is included here[12].

Aphasia due to lesions that isolate the perisylvian language areas from the other parts of the cerebral cortex – transcortical aphasia is also included here.

Eventhough anatomic basis is poorly defined, this emphasizes the importance of afferent, intercortical and efferent connections of language mechanisms. These are useful as the more common types of aphasia in revealing the complexity of language functions.

The locale of the lesion that causes loss of a language function does not localize the language function itself. This idea was put forth by Hughlings Jackson.

SUBCORTICAL APHASIA

In subcortical aphasia, the lesion is in the basal ganglia or deep cerebral white matter. Left thalamic haemorrhages produce a Wernicke like fluent aphasia with better comprehension than cortical Wernicke's aphasia[13].

Left basal ganglia and deep white matter lesions also cause aphasia. The lesion is an infarct involving the anterior putamen, caudate nucleus and anterior limb of internal capsule. They have dysarthria, decreased fluency, mildly impaired repetition and mild comprehension disturbance and less language dysfunction. This is anterior subcortical aphasia syndrome.

Posterior lesion involves the putamen and deep temporal white matter. They have fluent, paraphasic speech with impaired comprehension resembling Wernicke's aphasia. A large subcortical lesion involving both anterior and posterior lesion sides produce global aphasia. The insula a cortical structure shares a deep location with subcortical structures and it is associated with apraxia in aphasic patients.

3.3.3 Other Syndromes

a. Alexia and Agraphia

The oral language modalities of speech, naming, auditory comprehension and repetition are intact, but many cases manifest a fluent paraphasic speech with impaired naming. Associated deficits include right hemianopia and elements of Gerstmann's syndrome – agraphia, acalculia, right to left disorientation and finger agnosia. The lesion is in the inferior parietal lobule especially the angular gyrus.

b.Alexia without agraphia.

Pure word blindness is a rare but interesting form of reading disorder.It is visual equivalent of pure word deafness. In the classic form patients have no disorder of spoken language and can write normally but are not able to read. They have difficulty in object naming .Colour anomia and transient amnesia may be present. They have right homonymous hemianopia. Lesion is in left medial occipital lobe and splenium of corpus callosum.

c.Pure word deafness.

Patients are not able to understand spoken language despite normal hearing ability, oral expression, writing, reading aloud. The lesion is in left temporal lobe.

d.Aphemia.

It is called as pure word dumbness or cortical anarthria. The patient loses ability to speak but writes fluently. Reading and comprehension are intact. Right hemiparesis may be present. Aphemia follows a pure broca's area lesion.

e.Aphasic alexia.

Four patterns have been recognized.

- Letter by letter dyslexia is equivalent to pure alexia without agraphia.
- Deep dyslexia is a severe reading disorder in which patients recognize and read aloud familiar words especially nouns and verbs.
- Phonological dyslexia is similar to deep dyslexia with poor reading of nonwords. They read words without understanding.
- Surface dyslexia involves spared ability to read laboriously and inability to recognize words at a glance.

3.3.4 Other cerebral disorders of language.

Pathologic lesions that occur in the border zones between major cerebral arteries isolate perisylvian areas from other parts of the cerebrum and secondarily affect language function.

The lesions in the superior and lateral parts of frontal lobes impair all motor activity, to the point of abulia or akinetic mutism. The mute patient emits no sounds, in contrast to aphasic patients.

Extensive occipital lesions impair reading and also reduce the utilization of all visual stimuli.

Deep cerebral lesions, by causing inattention and disorientation, induce fragmentation of words and phrases and sometimes protracted, uncontrollable talking – logorrhea.

3.4 LOCALISATION OF CORTICAL LANGUAGE AREAS.

There are 4 main language areas, situated in most persons, in the left cerebral hemisphere. The entire language zone that encompasses these areas is perisylvian, bordering the sylvian fissure.

One, subserving the perception of spoken language, occupies the postero-superior temporal area (the posterior portion of area 22) and Heschl's gyri (areas 41 and 42), the posterior part of area 22 in the planum temporale is referred to as Wernicke's area.

The second area, subserving the perception of written language, occupies the angular gyrus (area 39) in the inferior parietal lobule, anterior to the visual receptive areas.

The supra marginal gyrus, lies between the auditory and visual language centers and the inferior temporal region, just anterior to the visual association cortex are part of central language zone.

The main executive region, situated at the posterior end of the inferior frontal convolution (areas 44 & 45) is referred to as Broca's area and is concerned with motor aspects of speech.

Visually perceived words are given expression in writing thru a fourth language area, the so called Exner writing area in the posterior part of second frontal convolution.

Sensory and motor areas are connected by network of nerve fibers, the arcuate fasciculus which passes through the isthumus of the temporal lobe and around the posterior end of sylvian fissure.

The visual receptive and somatosensory zones are integrated in the parietal lobe, and the auditory receptive zones in the temporal lobe.

The short association fibers join Broca's area with the lower rolandic cortex, which inturn, innervates the speech apparatus of the muscles of the lips, tongue, pharynx and larynx[14].

The Exner writing area is similarly integrated with the motor apparatus for the muscles of the hand.

The perisylvian language areas are also connected with the striatum and thalamus and with corresponding area in the minor, non dominant cerebral hemisphere through the corpus callosum and the anterior commissure.

The lesions that cause aphasia are usually in left cerebral hemisphere. This is because the left hemisphere tends to be dominant for language in both right handed and left handed people. In 90% of right handers and 60% of left handers the left hemisphere is dominant[15]. In people with right sided dominance, aphasia will develop with lesions in either the left or right hemisphere[16.]

Crossed aphasia is aphasia due to right hemispherical lesion in a right handed person[17].

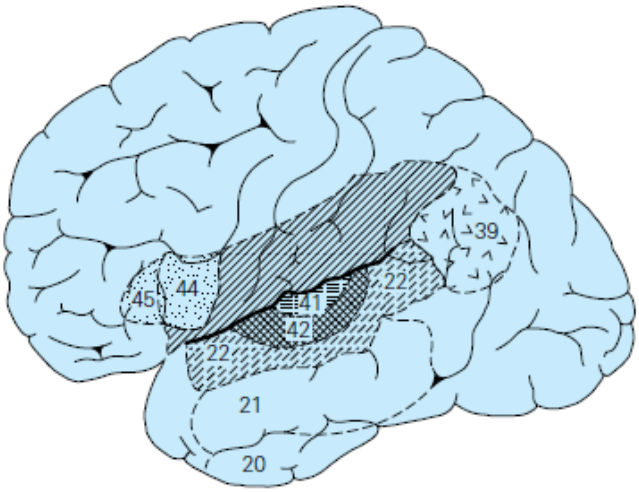


Figure 1. Schematic diagram showing cortical language areas

Broca's area is present in pars opercularis of the third frontal convolution(Broadman's area 44).Patients with motor aphasia have a lesion extending well beyond this area. The location and extent of lesion is important for prognosis. The patients with lesions limited to this area have a good recovery.

Wernicke's area is located in posterior part of left superior temporal gyrus (Broadman's area 22).Usually here the lesions extend beyond this area as a rule. Mostly temporal infarcts causing Wernicke's aphasia are due to embolism[18].

3.4.1 CT scan studies:

Margaret Nasser and Robert Hayward(1978)[19] in their study of aphasic patients with CT scan concluded that in motor aphasia, lesions were large involving both cortical and subcortical areas.

In Wernicke's aphasia the lesion is in temporal lobe affecting both cortical and subcortical areas.The supra marginal and angular gyrus of parietal lobe are also involved[20].

This temporo parietal lobe involvement is compatible with radionuclide brain scan[21] findings and with Yarneil's CT scan[22] finding in fluent aphasic patients.

Patients with conduction aphasia have deep lesions involving the arcuate fasciculus. This is compatible with Kertesz [21] radio nuclide brain scan study.

Transcortical motor aphasia is associated with dominant frontal lobe lesions that are anterior or superior to but do not include broca's area.

Global aphasia is associated with large lesions involving entire perisylvian region.

Large lesions are found in cortical and subcortical areas of frontal, parietal and temporal lobes.Kertesz[21] and Yarneil[22] also found extensive lesions in these areas in global aphasia.

In 1975, 30 patients were studied by Mohr et al[23] with autopsy and arteriogram. Lesion affecting broca's area causes mutism gradually replaced by rapidly improving dyspraxia and effortful articulation with no significant disturbance in language function. The complex of broca's aphasia is associated with larger infarct involving operculum, broca's area, insula and nearby cerebrum supplied by upper division of left middle cerebral artery.

3.4.2 Radionuclide Brain Scan Studies

Benson (1967)[13] studied 50 aphasic patients and reported successful correlation between lesion sites as localized on radionuclide brain scans. Fluent aphasia are associated with more posterior lesions and nonfluent aphasias are associated with more anterior lesions.

Kertesz, Ghent and Poole[21] studied 65 patients and found good correlation between the lesion sites as localized by scan. In addition to the non-fluent and fluent dichotomy, they found that patients with conduction aphasia had central lesions in relation to anterior posterior diameter. Most patients with anomia had parietal lobe lesions and most patients with global aphasia had extensive lesions involving more than 1 lobe. Using angiogram, radionuclide Brain scan and CT scan, Yarneil, Monroe and Sobel[22] had similar results.

3.5 APHASIA RECOVERY PATTERN

In general, recovery from aphasia due to cerebral trauma is usually faster and more complete that that from aphasia due to stroke.

The type of aphasia and its initial severity (extent of lesion) clearly influence recovery. Global aphasia usually improves very little and the same is true of severe Broca's and Wernicke's aphasias (Kertesz and Mccabe) [24.]

Characteristically, in the course of recovery, one type of aphasia may evolve into another type – global into severe Broca's, Wernicke's, Transcortical and Conduction into anomic aphasia.

Generally recovery from aphasia is most likely to occur from three to six months after stroke. Improvement continues for an indefinite period depending on patient's health, age, motivation and severity of the stroke. Greatest recovery is seen in the first three months. Young patients show recovery over a prolonged period of time[25].

Various types of aphasia recover differently. Zangwill and Butfield (1946) claimed that expressive aphasics improve most, while Vignolo (1964) noted that expressive disorders have a poor prognosis.

The studies of Butfield and Zangwill (1946),Wepman (1951)and Luria (1970)[26] indicated that post traumatic patients recovered better than stroke people Mark, Taylor and Rusk-1957,Godfrey and Douglass-1959 .

Vignolo in 1964 made first study of therapy to include the objective assessment of untreated patients. The comparison between 42 treated and 27 untreated patients yielded no statistical differences. Persistent anarthria and oral apraxia carried negative prognosis. Silverman, Sarno and Sands in 1970[27] reported a study of 31 global aphasics three months post onset to eliminate effects of spontaneous recovery. They were divided into three groups programmed instruction, non programmed instruction and no treatment.There was no difference in recovery between treated and untreated patients.

Sarno and Levita in 1979[28] studied spontaneous recovery in 14 patients using a subjective, functional assessment of language at two days, three and six months after stroke. They concluded that greatest change occurred in the first three months. Age, education or initial performance failed to correlate with the change. . In the first 6 months after stroke, fluent patients improved more than non-fluent and in the second 6 months, global aphasics improved most and fluent aphasics least. Improvement was noted in all aphasics on auditory comprehension task.

Andrew Kertesz and Patricia McCabe (1977)[24] studied spontaneous recovery in ninety three aphasics using western aphasia battery. Recovery rates was determined by measuring aphasia quotient. Maximum recovery was seen in motor aphasics, followed by conduction group. Anomic aphasia appeared to be a common end stage evolution. Long term follow up demonstrated that global aphasias have poor prognosis, Broca's and Wernicke' s have intermediate one.

Vijaya ragavan V and Natarajan V et al (1989)[29] studied recovery of language functions in strokes with aphasia in 16 patients using a modified form of western aphasia battery. In their study ,global aphasia showed significant overall improvement at the end of 8 weeks with significant improvement in verbal comprehension, repetition, and spontaneous speech.

Eshinger PJ, Damasio AR (1981)[30] studied age and gender of patients with different types of aphasia. Regardless of gender, patients with Broca and conduction aphasias were significantly younger than those with Wernicke and global aphasias. Reasons could be due to changes in stroke loci with age, cumulative effects of mental decline and age related changes in anatomic and physiologic mechanisms underlying language function.

Lendrem W, Lincoln NB(1985)[31] studied spontaneous recovery of language in patients with aphasia and showed that age, sex and aphasia type were not related to amount of improvement.

Functional imaging methods allow mapping of brain areas at work while performing language function, so possible to determine in aphasics patients the similarities and differences from normal activation pattern[32].

Studies have been published on the topic of neural basis of language[33] operations and neuro biology of recovery[33-37]. The results are divergent, because the patients belonged to different aphasic pattern, but also because some used group averaging methods[33] , where as others studied single cases[34-38].

Studies of language activation, PET, SPECT, scanning techniques advance our understanding of neuroanatomy of language recovery.(Heiss et al, 1999)[39].

Studies have revealed that there is excessive activation of homologous right sided brain regions compared with normal subjects. Some attributed this to recovery[33-35,] others see it as an expression of abnormal function predicting poor recovery[37].

Studies of Ohyama M, Senda M ,Kitamura S[34] and studies of Heiss WD, Karbe H, Weber Luxenburger G[37], prove that preserved left hemisphere perilesional tissue subserve the return of function.

Longitudinal studies[37] correlate the changes in brain activation pattern over time with parallel changes in performance in language operations and shed light about meaning of differences in brain activation pattern. Speech reorganization is a highly dynamic process involving the establishment of new communications in the remaining system.

Improving the perfusion adjacent to the lesion is crucial for recovery. The coactivation between cerebral representations of hand movements and language can be used therapeutically for aphasia.

Intensive and specific language therapy interact with brain plasticity to accelerate the process of recovery. Neuro imaging studies suggest , recovery from aphasia is due to activation of right hemisphere areas and preserved perilesional left hemisphere areas such as frontal lobe and basal ganglia resulting in slow and gradual compensatory functions.

In the acute phase after stroke, right inferior frontal gyrus gets activated whereas in the chronic phase left frontal lobe activation improves speech production and left temporal lobe activation improves speech comprehension. Agrammatic aphasics retain gap filling capacity and treatment improves their ability to make use of this capacity.

3.6 SPEECH THERAPY AND REHABILITATION.

Language recovery is mediated by shifting of functions to right hemisphere or due to adjacent left hemisphere regions[39]. Trials indicate patients who undergo formal speech therapy recover better than untreated patients. (Robey, 1998)[40.]

Language rehabilitation can be done by using pharmacological agents. Dopaminergic drug bromocriptine promotes expressive speech out put in transcortical aphasia. (Albert , 1998). Stimulant drugs can also be used.

Speech therapy is usually provided by speech language pathologists. They facilitate language recovery by a variety of techniques and help the patient to compensate for lost function. Repeated practices in articulation and comprehension stimulate improvement.

Stimulation – Facilitation techniques for aphasia therapy.

- Gestural expression and pointing
- Word to picture matching
- Yes- no response reliability
- Oral – motor imitation
- Phoneme, then word repetition
- Verbal cueing for words, sentence completion
- Auditory processing at phrase level and then sentence level
- Word, phrase, then sentence level reading
- Graphic talks, tracing, copying, word completion
- Conversational skills
- Psychological and social supports

Commonly ten hours of speech therapy is given weekly for at least six weeks.

Other treatment methods :

Melodic intonation therapy uses melody to involve right hemisphere in speech production[4]. Visual action therapy uses gestural expressions. Treatment of aphasic perseveration reduces repetitive utterances. Functional communication therapy utilizes the advantage of extra linguistic communication. cVIC or Lingraphica are computer programmes developed for primate communication[4]. Patients who cannot speak can learn to produce simple sentences via computer. Augmentative devices make language expression possible through use of printers or voice simulators.

Pharmacological agents used in speech therapy.

- Piracetam
- Cholinergic agents
- Stimulant drugs – Amphetamine
- Dopaminergic agents -Bromocriptine

Piracetam is a derivative of GABA[4], but with no GABA activity. It increases cholinergic and aminergic transmission. It also increases cerebral blood flow in left hemisphere language regions.

MATERIAL AND METHODS

4.1 STUDY DESIGN

Cross sectional prospective study.

4.2 STUDY POPULATION

Patients who developed aphasia with or without other neurologic deficits due to acute stroke admitted in Coimbatore Medical College Hospital, Coimbatore during the period July 2007 to June 2008 were taken up for study after getting informed consent. This study was approved by the Ethics Committee of our College, Coimbatore. Consent form enclosed.

4.3 INCLUSION CRITERIA

The patients taken up for the study were

1. Right handed persons fulfilling the criteria for handedness.
2. Patients with lesion in left hemisphere as confirmed by CT scan.
3. Patients with tamil as mother tongue.
4. Patients with normal hearing threshold
5. Patients coming for regular follow up

4.4 EXCLUSION CRITERIA

Patients were excluded from the study if they had

1. Pre existing language or speech disorder.
2. Psychiatric disease or previous stroke.
3. Aphasia secondary to head trauma, tumor.
4. Equivocal handedness.
5. Evidence of right hemisphere lesion.

4.5 METHODS OF STUDY

A detailed history was taken and a complete clinical examination was done. Blood biochemical analysis, hemogram, ECG, Echocardiogram were done. CT scan brain was taken.

The bedside analysis[4] of aphasic disorders entails the systematic testing of six aspects of language function:

1. Conversational speech (fluency)
2. Comprehension
3. Repetition
4. Reading
5. Writing
6. Naming

Language function was assessed by tamil version of modified western aphasia battery system at the end of 4th week (T1) and repeated at 8th week (T2) 16th week (T3) and at 24th week (T4) with a variation of plus or minus one week.

4.5.1 Test battery and scoring system.

The assessment of aphasia is done by various techniques. Specific assessment instruments will show a good deal of variability. Matching the assessment to any given patient requires that the clinician maintain a flexible and knowledgeable manner of dealing with the task examining aphasia. This manner of examination is core to what it means work as a clinician, rather than testing patients as a technician or as a gatherer of research data (e.g., Matarazzo, 1990). The test battery used to assess the language function is tamil version of modified western aphasia battery (Kertesz and Poole 1974)[41]. In this four language parameters namely spontaneous speech, auditory comprehension, repetition and naming were tested and scored and final aphasia quotient arrived as discussed below.(Protocol)

1. Spontaneous speech

Fluency and information content were tested in speech assessment. This was tested by conventional questions and patient is given a simple picture to describe. Carefully graded criteria were used to judge the fluency in a 1-10 scale (Table 2). It was scored for information content, depending on the number of items, correctly answered and 1 -10 score was allotted (Table 3). So a total score of 20 was obtained for spontaneous speech.

SCORING OF SPONTANEOUS SPEECH

TABLE 2: FLUENCY, GRAMMAR AND PARAPHASIAS.

0. No response.
1. Meaningless utterances.
2. Utterances are used with inflection of language.
3. Occasional correct word.
4. Telegraphic sentences.
5. Moderate fluency, a few words together.
6. Predominantly sentences.
7. Fluent jargon.
8. Circumlocutory fluent speech.
9. Slight word finding difficulty.
10. Normal fluency without hesitation.

TABLE 3: INFORMATION CONTENT

0. No information.
1. Incomplete responses only
2. Correct response to any 1 item.
3. Correct response to any 2 items.
4. Correct response to any 3 items.
5. Correct response to any 3 of the first 6 items plus some response to the picture.
6. Correct response to any 4 of the first 6 items plus some response to the picture.
7. Correct responses to any 4 of the first 6 items and a mention of at least 6 of the items in the picture.
8. Correct responses to 5 of the first 6 items;incomplete description of picture.
9. Correct responses to all 6 items.An almost complete description of picture
10. Correct responses to all the 6 items and to the picture.Sentences of normal length and complexity.A reasonably complete description of the picture.

2.Auditory verbal comprehension.

Comprehension was measured in three ways, first the patient was asked yes or no questions of graded complexity. Three marks were given for correct answer. Twenty questions were asked and sixty marks scored for that. Then the patient is asked to point 6 different real objects, drawn objects, forms, letters, numbers, furnitures, body parts, fingers and colours. Patient was asked to show three left and right sided parts. One mark was allotted for each item and total score of sixty was arrived.(Protocol).

Finally the patient to perform sequentially ordered auditory commands with three objects pointing to each other or placing in relation to other.A total score of eighty was allotted.According to complexity of commands, the score was given.(Protocol.)

The total subscores was summed up,divided by 20 and final score was arrived. (Final max. score 10.)

3. Repetition.

Repetition was tested with words, numbers and increasingly complex sentences. Marks were allotted according to the words and sentences.This was divided by 10 and final maximum score was calculated for 10.

4. Naming

Naming was tested by

a. Asking the patient to name 20 objects.3 marks was allotted for each objects and a total score of 60 was allotted.

b. Asking the patients to name the animals for 1 minute. A maximum score of 20 was allotted.

c. Asking the patient to complete a sentence.5 sentences were given and maximum score of 10 was given.

d. Asking the patient to answer 5 questions ,in a single word response.A maximum score of 10 was given.

Finally, all scores were summed up and divided by 10 and a final maximum score 10 marks was calculated.(Protocol.)

Aphasia quotient(AQ),maximum score -100 was obtained by summing up the subscores, max. score 50 and multiplied by 2.This test battery was applied to the patient at each test intervals and aphasia quotient was calculated and compared. Reading,

writing, calculation and drawing were not tested.

4.5.2 Definition of population.

Aphasia are subdivided into groups according to initial subscores(Table 4a). Fluency divides global, broca, transcortical motor aphasics from wernicke, conduction and transcortical sensory aphasics. Comprehension separates broca from global, wernicke from anomic, conduction aphasia. Repetition splits transcortical, conduction aphasia from other aphasias.

TABLE 4.a
CRITERIA FOR CLASSIFICATION OF APHASIA

	Fluency	Comprehension	Repetition	Naming
1. Global	0-4	0-3.9	0-4.9	0-6
2. Broca	0-4	4-10	0-7.9	0-8
3. Wernicke	5-10	0-6.9	0-7.9	0-7
4. Conduction	5-10	7-10	0-6.9	0-9
5. Anomic	5-10	7-10	7-10	0-9
6. Transcortical Motor	0-4	4-10	8-10	0-8
7. Transcortical Sensory	5-10	0-6.9	8-10	0-9

Prognostic grading of various aphasias were done by using test scores.(Table 4b)

TABLE 4.b
PROGNOSTIC GRADING OF VARIOUS APHASIAS AND TEST SCORES

Grade	Test Scores
Poor	0-25
Fair	26-50
Good	51-75
Excellent	76-100

4.5.3 Identification of cortical language areas in CT scan.

CT scan brain was taken with a plain of each section angled 20 degrees to the cantho meatal line. The corresponding CT slices were labeled sequentially from the base towards the vertex according to the known cortical language areas present in each slice. Broca, Wernicke, supramarginal and angular gyrus areas on these brain slices have had easily identifiable relationships to the specific parts of the ventricular system as discussed below.

1.Broca's area

It is present in the frontal lobe, lateral to the inferior portion of the anterior horn of the left lateral ventricle. The cortical representation is present lateral to the anterior horn of the left lateral ventricle.

2.Wernicke's area.

This area 22 is present, lateral to the third ventricle and quadrigeminal cistern in the temporal lobe.The area is lateral and just anterior to the atrium of the left lateral ventricle.The density of the calcified choroid plexus in the atrium serves as anatomic land mark relative to Wernicke's area on CT scan brain.

3.Supra marginal and angular gyrus areas.

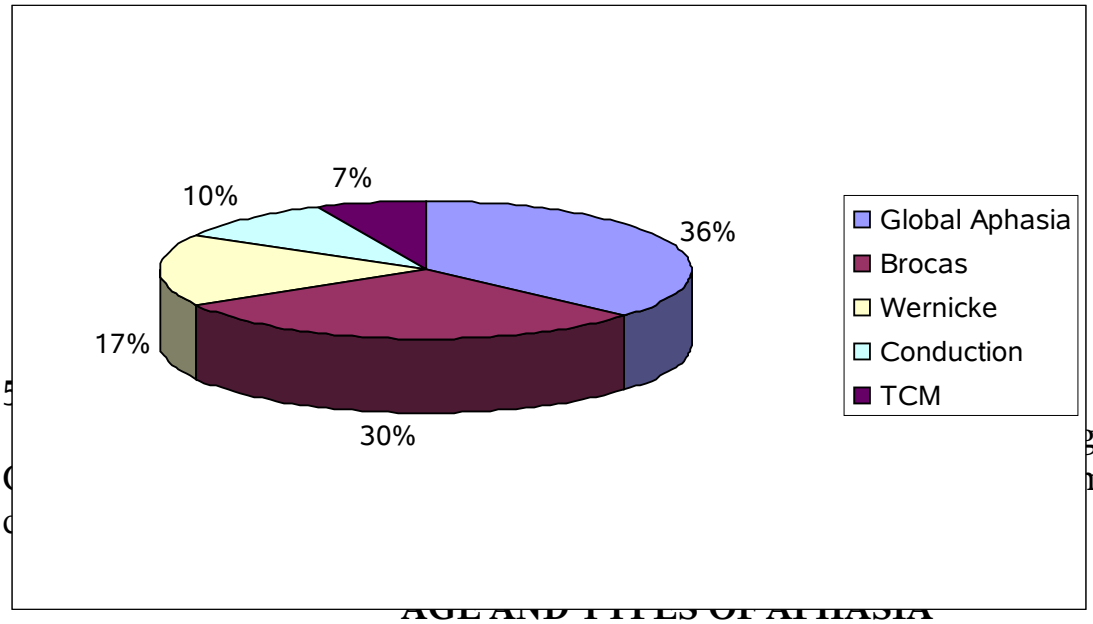
The cortical areas 40 and 39 are usually observed,lateral to the posterior portions of the body of the left lateral ventricle.

5. RESULTS AND ANALYSIS

5.1 NUMBER OF PATIENTS

Of the 90 stroke patients admitted to our ward during the study period 30 cases were selected in accordance with inclusion criteria based on language, education etc. Of the 90 stroke patients, 18 lost to follow up, 19 died and 23 were strokes due to other causes (trauma, tumor). Two cases of embolic stroke due to preexisting rheumatic heart disease also died within 2 weeks of admission and could not be followed up for aphasia recovery pattern.

Out of this global aphasics were 11(n=11). Broca's aphasics were 9(n=9),Wernicke's aphasics were 5(n=5), conduction aphasics were 3(n=3) and transcortical motor aphasics were 2(n=2).



ge was 50 years.
mited number of

AGE AND TYPES OF APHASIA

Age	N	
30-40	7 (23%)	5
41-50	11(36%)	2
51-60	7(23%)	3
61-70	5(16%)	1

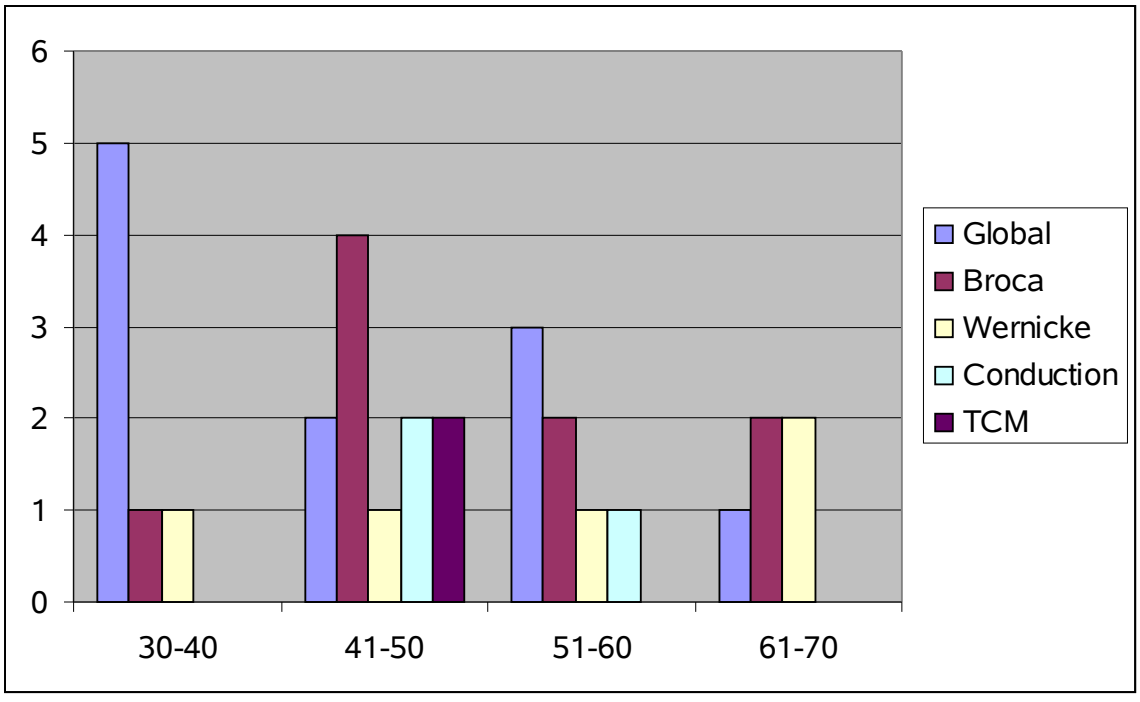


Figure 3. Age wise

distribution of aphasias

5.3 SEX

25 males and 5 females were included in the study.Out of 25 males ,9 suffered from global aphasia,9 from Broca's aphasia, 3 from Wernicke's aphasia,2 from conduction aphasia and 2 from transcortical aphasia. Out of 5 females,2 suffered from global aphasia,2 had Wernicke's aphasia and 1 had conduction aphasia.Because of limited cases ,sex and recovery pattern could not be compared.

TABLE 6

SEX AND TYPES OF APHASIA.

Sex	N	Global	Broca	Wernicke	Conduction	TCM
Male	25(83%)	9(36%)	9(36%)	3(12%)	2(8%)	2(8%)
Female	5(16%)	2(40%)	0	2(40%)	1(20%)	0

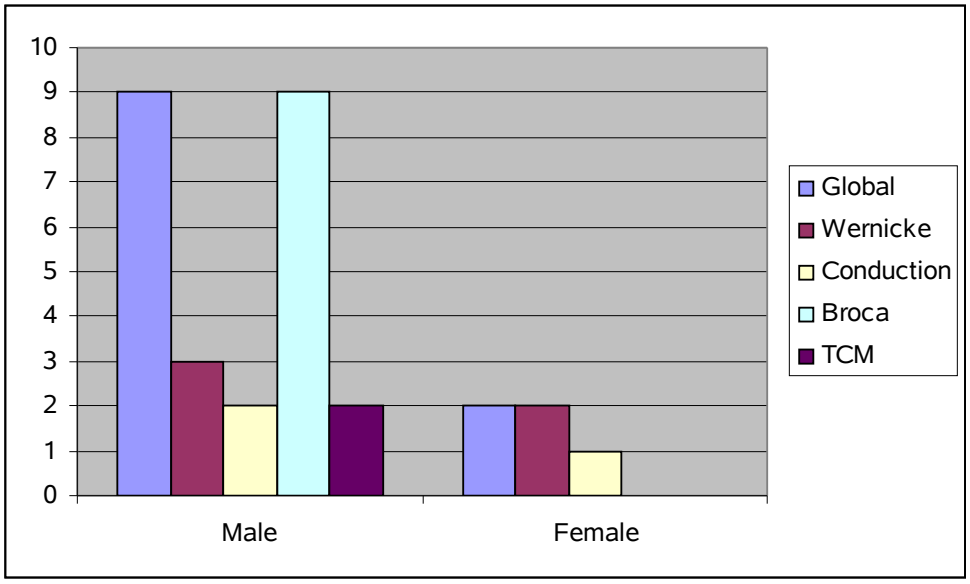


Figure 4. Sex distribution of aphasias

5.4 EDUCATION

Out of 30 patients,1 patient studied upto 10^{th} std,3 upto 8^{th} std,2 upto 6^{th} std,5 upto 5^{th} std,7 patients upto 4^{th} std,2 upto 3^{rd} std and 10 patients were illiterate. There was no significant difference in recovery among literate and illiterate patients**.**

5.5 RISK FACTORS FOR STROKE

Several risk factors have been identified to influence the recovery from aphasia. M. T. Sarno and Levita(1971) reported that aphasic individuals who were employed at the time of stroke recovered more than those who were unemployed. The presence of depression, anxiety, and paranoia have been cited as negative factors in recovery (Benson, 1979a, 1979b, 1980; Damasio, 1992;

Lebrun, 1980; M. T. Sarno, 1993). Premorbid traits have been identified as important prognostic factors (Eisenson, 1973; Herrmann, Britz, Bartels, & Wallesch, 1995; Wepman, 1951). Eisenson (1949, 1964, 1973) felt that patients with outgoing personalities had a better prognosis than those with introverted, dependent, or rigid personalities.

5.5.1 Diabetes.

Out of 30 patients,10 had diabetes.Out of 10,3 developed global aphasia,2 Broca,3 Wernicke which showed poor recovery and 2 patients developed conduction aphasia and showed good recovery.

5.5.2 Hypertension

Out of 30 patients, 14 patients had high blood pressure as a risk factor. Out of 14,5 patients developed global aphasia,3 patient developed Broca,3 patients developed Wernicke, 2 had conduction aphasia and 1 patient developed trans cortical motor aphasia.

5.5.3 Smoking

Out of 30 patients, 22 patients were smokers. 8 developed global aphasia, 9 developed broca's aphasia, 2 developed wernicke's aphasia, 2 had conduction aphasia and 1 developed transcortical motor aphasia.

5.5.4 Alcoholism

Out of 30 patients, 6 patients were alcoholics.4 patients developed broca's aphasia,1 developed wernicke's aphasia and 1 patient developed global aphasia.

TABLE 7
RISK FACTORS OF STROKE AND TYPES OF APHASIA.

Risk factor	N	Global	Broca	Wernicke	Conduction	TCM
HT	14	5	3	3	2	1
DM	10	3	2	3	2	0
Smoking	22	8	9	2	1	2
Alcoholic	6	1	4	1	0	0

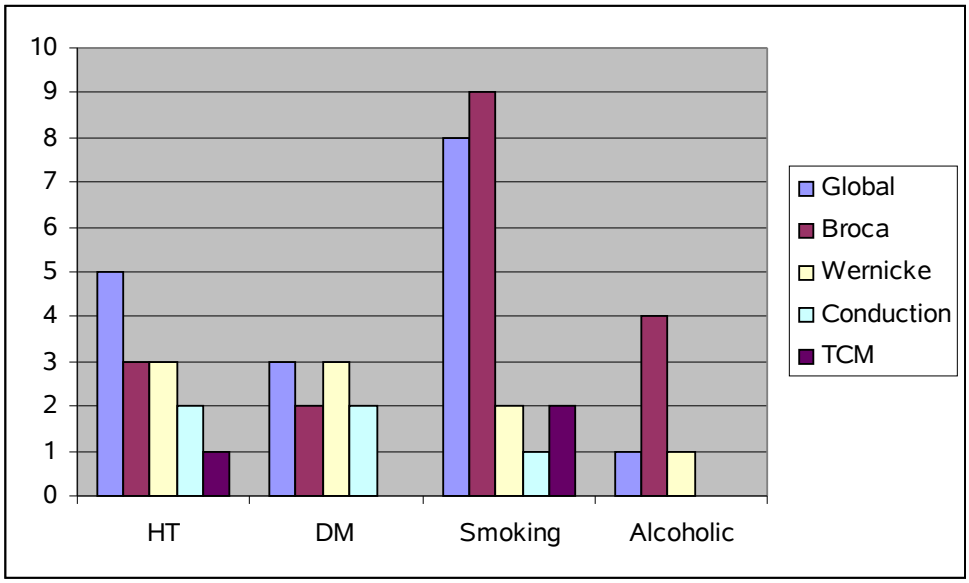


Figure 5. Risk factors among aphasic patients

5.6 TYPES OF APHASIA AND RECOVERY RATES

5.6.1 Global aphasia.(n=11).

These patients showed limited recovery in 6 months period.However,3 of them showed good improvement and evolved into broca's aphasia.

TABLE 8
GLOBAL APHASIA AND TEST SCORES

Case No.	T1 (4wks) (%)	T2 (8wks) (%)	T3 (16wks) (%)	T4 (24wks) (%)	Initial type of aphasia	Final evolution
1	2.8	3.6	4.2	4.2	Global	Global
2	2.2	2.4	2.6	2.6	Global	Global
3	2.2	3.6	4.0	4.0	Global	Global
4	3.0	8.0	16.0	18.0	Global	Global
5	4.8	5.6	8.6	10.0	Global	Global
6	2.6	3.8	6.2	9.8	Global	Global
7	8.8	36.4	48.0	62.0	Global	Broca
8	13.4	38.6	54.0	60.0	Global	Broca
9	13.6	38.8	56.0	64.0	Global	Broca
10	4.8	5.8	8.8	12.0	Global	Global
11	4.8	5.6	8.6	12.0	Global	Global

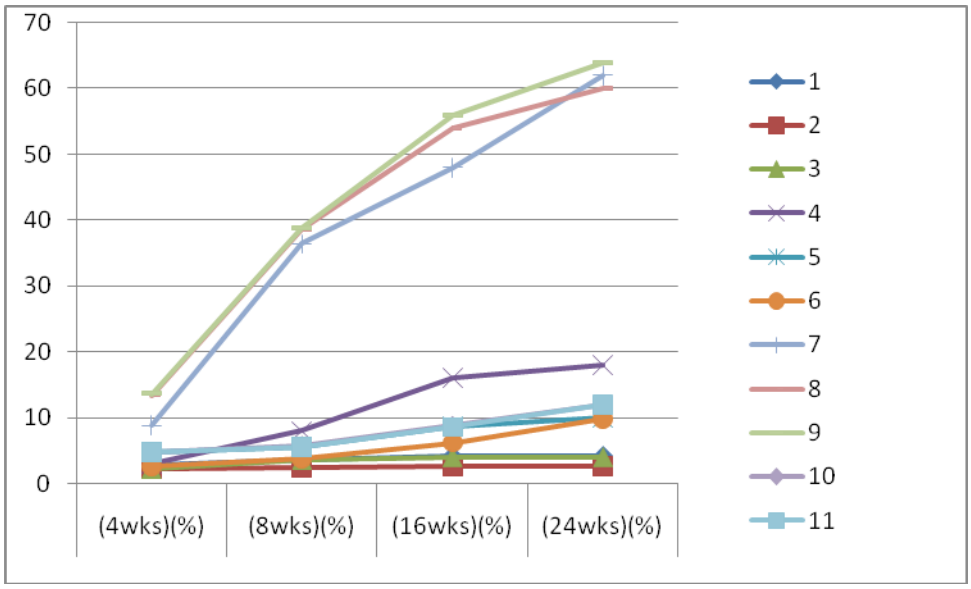


Figure 6: Evolution of global aphasia

5.6.2 Broca's aphasia(n=9)

These patients showed overall recovery in all test period.2 of them evolved into transcortical motor aphasia.2 did not show significant improvement.

TABLE 9

BROCA'S APHASIA AND TEST SCORES

Case No.	T1(4 Weeks) %	T2 (8 Weeks) %	T3 (16 Weeks) %	T4 (24 Weeks) %	Initial type of aphasia	Final Evolution
12	60.2	70.4	70.8	95.2	Broca	TCM
13	36.0	48.0	49.0	52.0	Broca	Broca
14	26.0	33.2	42.2	50	Broca	Broca
15	21.0	23.0	25.0	28.0	Broca	Broca
16	56.4	65.2	70.0	90.4	Broca	TCM
17	22.0	31.0	40.0	50.0	Broca	Broca
18	21.0	23.0	25.0	28.0	Broca	Broca
19	22.0	31.0	42.0	52.2	Broca	Broca
20	22.0	33.0	46.0	54.0	Broca	Broca

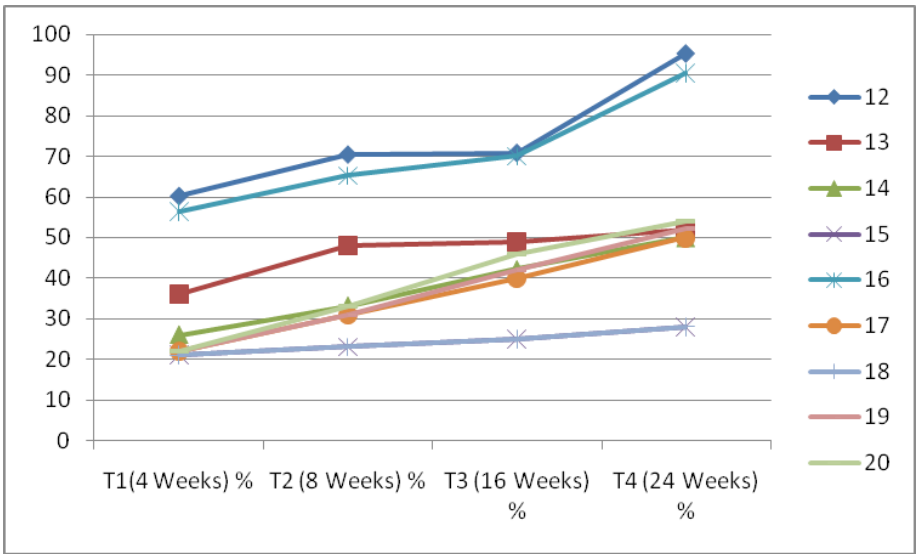


Figure 7 : Evolution of Broca's aphasia

5.6.3 Wernicke's aphasia(n=5)

These patients did not show much improvement throughout the test period and remained as wernicke's aphasia at the end of 6 months period.

TABLE 10

WERNICKE'S APHASIA AND TEST SCORES

Case No.	T1(4 Weeks)%	T2(8 Weeks)%	T3(16 Weeks)%	T4(24 Weeks)%	Initial type of aphasia	Final evolution
24	24.6	35.0	36.0	36.0	Wernicke	Wernicke
25	20.0	26.0	26.8	34.0	Wernicke	Wernicke
26	20.0	26.2	26.8	36.0	Wernicke	Wernicke
27	22.0	26.0	26.0	28.0	Wernicke	Wernicke
28	20.0	28.0	32.0	36.0	Wernicke	Wernicke

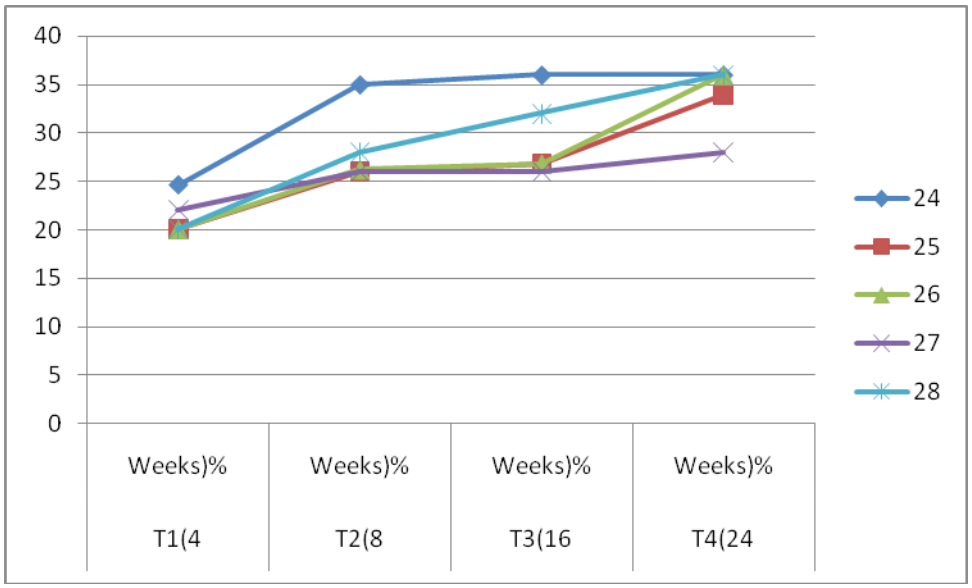


Figure 8: Evolution of Wernicke's aphasia

5.6.4 Conduction aphasia(n=3)

These patients showed favourable spontaneous recovery pattern.2 patients became anomic aphasia and another remained as conduction aphasia.

TABLE 11

CONDUCTION APHASIA AND TEST SCORES

Case No.	T1(4 Weeks) %	T2 (8 Weeks) %	T3 (16 Weeks) %	T4 (24 Weeks) %	Initial Type of Aphasia	Final Evolution
21	81.7	88.0	92.8	94.0	Conduction	Conduction
22	58.4	72.0	92.0	92.0	Conduction	Anomic
23	53.4	68.0	86.0	92.0	Conduction	Anomic

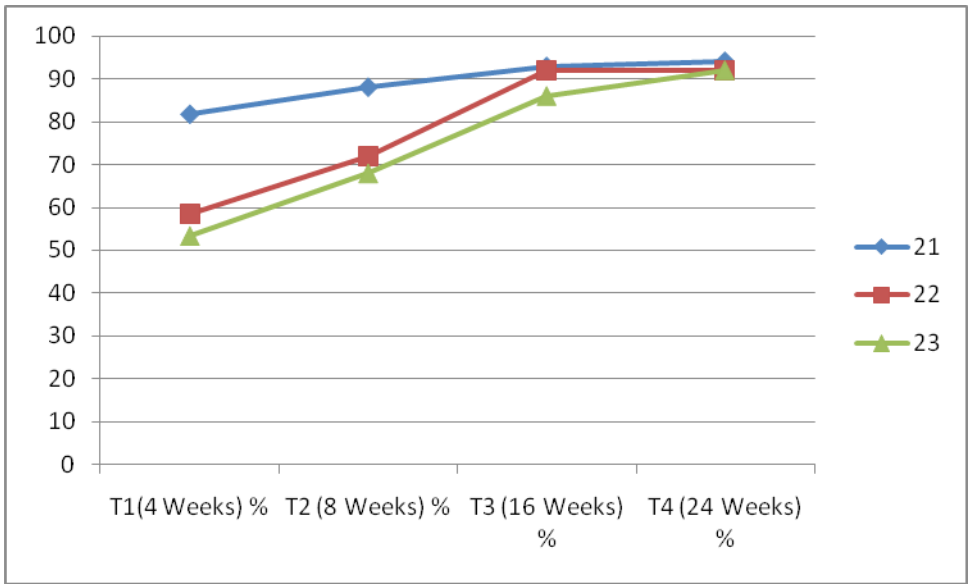


Figure 9: Evolution of Conduction aphasia

5.6.5 Transcortical motor aphasia(n=2)

These patients showed good improvement in test period and became normal.

TABLE 12
TRANSCORTICAL MOTOR APHASIA AND TEST SCORES

Case No.	T1(4 Weeks) %	T2 (8 Weeks) %	T3 (16 Weeks) %	T4 (24 Weeks) %	Initial Type of Aphasia	Final Evolution
29	50.0	64.0	78.0	92.0	TCM	Normal
30	50.0	66.0	80.0	94.0	TCM	Normal

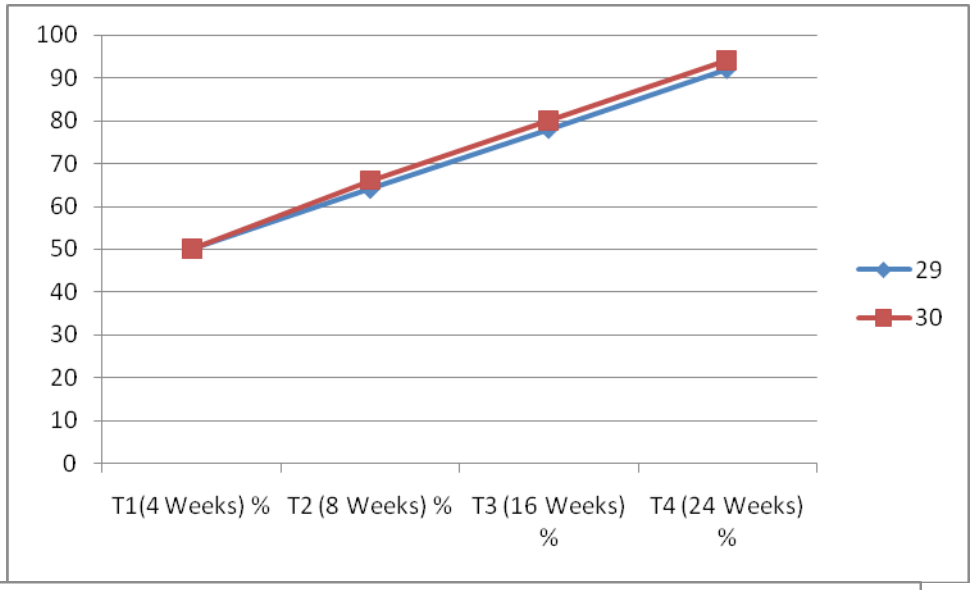


Figure 10: Evolution of Transcortical motor aphasia

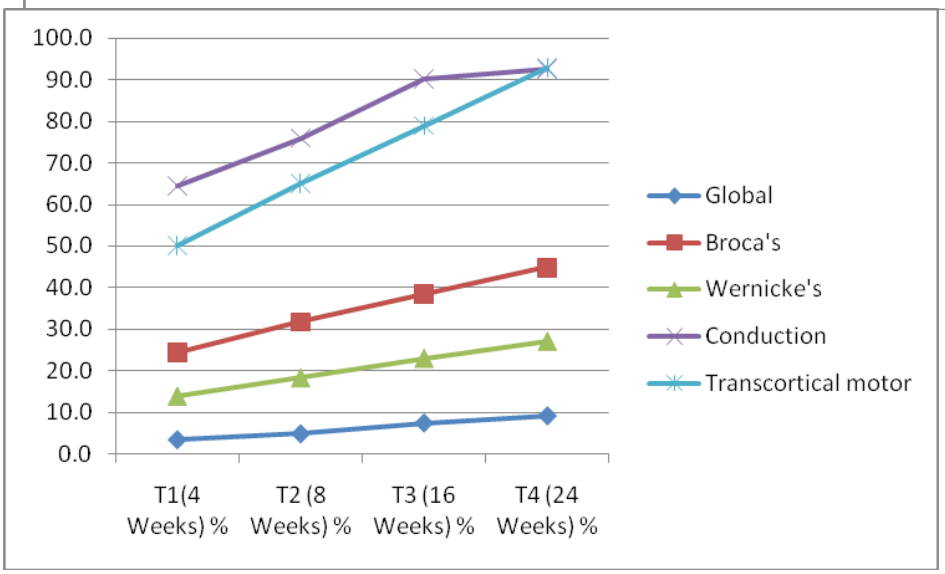


Figure 11: Comparison of evolution of aphasias

Comparison of various aphasias as shown in figure 11 shows that global aphasia shows the least

improvement as shown in previous studies. This is probably due to the more extensive involvement of cerebrum particularly the frontal operculum in global aphasias. Compared to wernicke's and broca's , broca's aphasia recovers much better than wernicke's, also is to note from the graph that lower the score at presentation lower is the extent of recovery as shown in previous studies.

5.7 PROGNOSIS OF VARIOUS APHASIAS

These 30 patients were followed for 6 months and outcome was evaluated. The aphasia quotient values of the last test were used to correlate the language performance at the end of follow up as poor, fair, good and excellent (Andrew Kertez and McCabe study -1977).

TABLE 13
PROGNOSIS OF VARIOUS APHASIAS

Aphasia type initially	N	Poor 0-25	Fair 26-50	Good 51-75	Excellent 76-100
Global	11	8	0	3	0
Broca	9	2	0	5	2
Wernicke	5	1	4	0	0
Conduction	3	1	0	0	2
Transcortical Motor	2	0	0	0	2

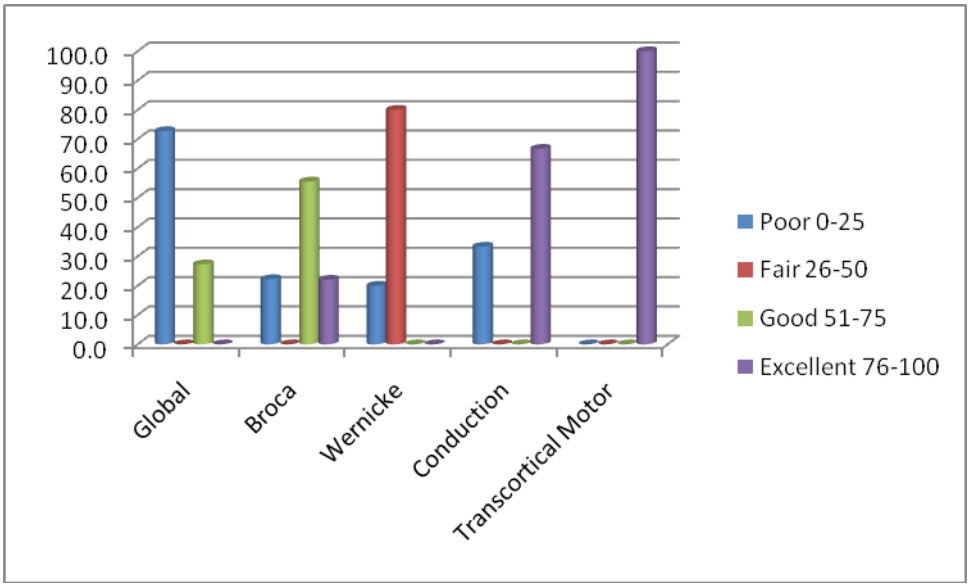


Figure 12: Prognosis of various aphasias.

5.8 OUTCOME AND INITIAL SEVERITY

Those patients who had low scores during initial examination recovered to lesser extent and who had high scores show good improvement. So the initial severity of aphasia and final outcome in 6 months was found to be significantly correlated.

TABLE 14

INITIAL SCORES AND OUTCOME OF VARIOUS APHASIAS

Type of Aphasia	N	Initial AQ (%) T1 (4 Weeks)		Final AQ (%) T4 (24 Weeks)		Outcome
		Range	Mean	Range	Mean	
Global	11	2.2 – 13.6	5.72	2.6 – 64.0	23.5	Poor
Broca	9	21.0 – 60.2	31.0	25.0 – 95.2	58.0	Good
Wernicke	5	26.0 – 24.6	21.0	28.0 – 36.0	33.0	Fair
Conduction	3	58.4 – 81.7	70.0	92.0 – 94.0	93.0	Excellent
TCM	2	50.0 – 52.0	51.0	92.0 – 94.0	93.0	Excellent

5.9 EVOLUTION OF APHASIA

Three of the global aphasics became broca's aphasia.2 of broca's aphasics became transcortical motor aphasia. In conduction aphasics 2 became anomic aphasia and one remained the same. Transcortical motor aphasics became normal.

TABLE 15

EVOLUTION OF APHASIA

Initial Aphasia	N	End Stage	N
Global	11	Global Broca	8 3
Broca	9	Broca TCM	7 2
Wernicke	5	Wernicke	5
Conduction	3	Conduction Anomic	1 2
TCM	2	Normal	2

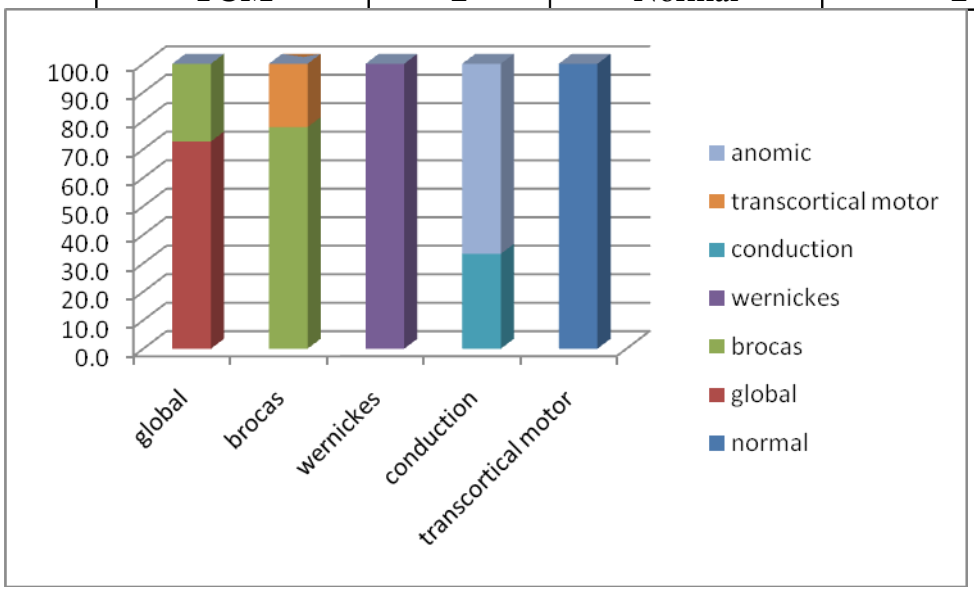


Figure 13 : Final evolution in various aphasias

5.10 CORRELATION WITH CT SCAN.

5.10.1 Global aphasia

All patients showed large infarct involving the broca's , wernicke's and adjacent areas. Large portions of left frontal, parietal and temporal lobes involved both cortically and subcortically.

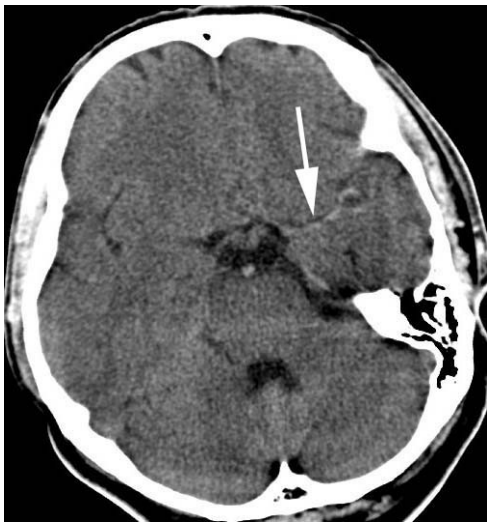


Figure 14. Dense MCA sign in a patient with global aphasia

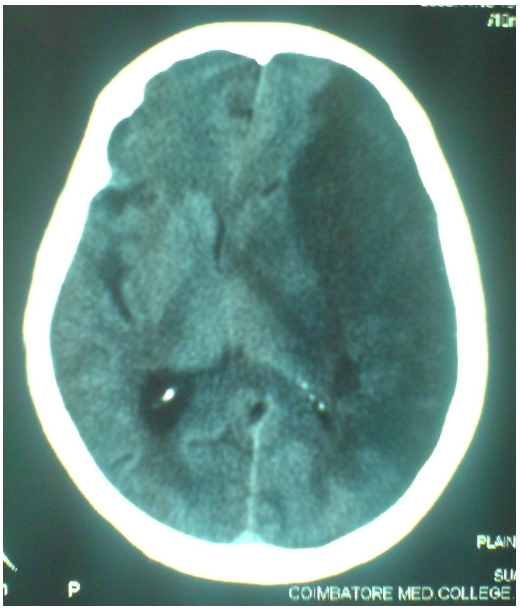


Figure 15. Large left MCA territory infarct with global aphasia.

5.10.2 Broca's aphasia

Here CT scan showed infarct in the left frontal lobe involving broca's area and sub cortical areas .Temporal lobe and wernicke's area were spared Both cortical and subcortical structures were involved.

Figure 16. Infarct left frontal lobe –Broca's aphasia

5.10.3 Wernicke's aphasia

Infarct was noted in left lower temporal lobe and involved wernicke's area.The supramarginal gyrus of the parietal lobe was involved. Broca's area not involved. Deeper structures were also involved.

Figure 17. Infarct left lower temporal lobe- Wernicke's aphasia

5.10.4 Conduction aphasia

Infarct was seen lateral to the posterior portion of the body of left lateral ventricle consistent with location of the posterior portion of the arcuate fasciculus.Wernicke's area spared. The lesion continued superiorly into upper parietal lobes. Both cortical and subcortical structures were involved.

5.10.5 Transcortical motor aphasia

Infarct was noted in left frontal lobe anterior and superior to broca's area.

6. DISCUSSION

Many studies have focused on recovery pattern of aphasia in stroke patients but there is no uniformity. The patients were not segregated on the basis of etiology of stroke. In our study we have focused only on ischemic and hemorrhagic stroke, excluding trauma, infections, degeneration, tumors and vasculitis. This study focused on the recovery pattern of aphasias in 30 stroke patients with western aphasia battery system.

The present study showed that among patients with broca's aphasia 2 out of 9 had excellent recovery, 5 had good recovery and 2 had poor recovery. Andrew Kertesz and McCabe(1977)[24],Weisenberg and McBrides (1935) showed good recovery in broca's aphasia as in our study.

Kertesz and McCabe (1977)[24] noted bimodal recovery in wernicke's aphasia, those with initially low scores did poorly and those with high scores improved well. In our study, all wernicke's aphasics had low initial scores and showed fair recovery. This pattern was seen among patients with global aphasia also. Sarno, Silverman and Sands(1970)[27] in their study noticed good outcome in those with high initial scores. Kertesz and McCabe (1977)[24] also noted this fact. In our study also there was good correlation between initial scores and outcome.

Patients with conduction and transcortical motor aphasia had excellent recovery. But with global aphasics 8 out of 11 showed poor recovery and 3 had good recovery. This was also noted by Kertesz and McCabe[24] in their study.

As shown in figure 13, three patients with global aphasia transformed into broca's aphasia, 2 patients of broca's aphasia turned into transcortical motor aphasia, 2 patients with conduction aphasia turned into anomic aphasia and 2 with transcortical motor transformed into nonaphasics. This observation was already noted by Andrew and McCabe[24] in their study.

Lendren W, Lincoln (1985)[31] studied spontaneous recovery of language in aphasic patients between 4 and 34 weeks which revealed age, sex and aphasia type were not related to amount of improvement. No significant sex difference in recovery was found in our study. It was observed that hypertensive and diabetics developed larger infarcts (global aphasia) and showed poor recovery.

Within the groups showing recovery, significant improvement was noted within eighth week of onset of stroke in our study. Sarno and Levita[28] also noted the improvement in first three months after stroke. In 1979 they used a subjective ,functional assessment of language at two days, three months and six months after stroke. They concluded that greatest change occurred in the first 3 months. Age and

education failed to correlate the changes. Culton's studies (1971) also supported this view.

Eslinger PJ, Damasio AR (1981)[30] studied the age and gender of aphasic patients. Regardless of gender, patients with broca's and conduction aphasia were significantly younger than those patients with wernicke and global aphasia[30,42-46]. In our study, no distinction between aphasia type in different ages was made because too few were in each group for meaningful correlation. When the mean ages of various types were compared no significant differences were apparent.

Comprehension tends to recover to a greater degree than expression(Basso, Capitani, Zonobio et al., 1982; Kenin & Swisher, 1972; Lebrun, 1976). In our study also patients with global and wernicke aphasia showed overall poor prognosis but considerable recovery was noticed in auditory verbal comprehension. Vijayaragavan and Natarajan et al[29] in their study also noted this observation.

Basso et al' found that global and Wernicke's aphasics responded to treatment of 6 months or more duration.

There was good correlation with the anatomical location of lesion and CT brain. Separate lesion sites for broca's, wernicke's, global and transcortical motor aphasias were demonstrated on CT brain. The lesion sites were consistent with Geschwind's concept of aphasia.

Patients whose computerized tomography scans show large dominant hemisphere lesions, many small lesions, or bilateral lesions are less likely to recover than those with smaller or fewer lesions (Kertesz, 1979; Yarnell et al., 1976). Lesions in Wernicke's area or those that extend more posteriorly tend to lead to severe and persistent aphasia (Ludlow et al., 1986;Mohr et al., 1978)

It was interesting to note that three patients with global aphasia which was due to hemorrhagic lesions recovered and turned into broca's aphasia and this could possibly be explained by the following fact. Haemorrhages compress cerebral tissue without destroying it, so recovery from aphasia is better in haemorrhagic strokes than in ischemic strokes although haemorrhages are more fatal[4].

Aphasias due to vascular lesions may be due to ischemic strokes – embolic and thrombotic lesions. Deficit due to embolism may be sudden and maximal at the onset. Deficit due to thrombosis gradually increases and may be waxing and waning[4]. Wernicke's aphasia[47] is mostly due to embolic lesion in the inferior division of left middle cerebral artery.

Global aphasia is due to embolus to MCA, thrombosis of ICA and may also be due to haemorrhage into deep basal ganglia due to hypertension[4]. Broca's aphasia is

mostly due to lesion in the superior division of left middle cerebral artery. Transcortical aphasia is mostly due to water shed zone infarct between anterior and middle cerebral arteries[11] and at times may be due to occlusion of anterior cerebral artery. Pure alexia without agraphia may be due to occlusion of posterior cerebral artery.

This suggests that cerebral hypoperfusion is an accurate indicator of aphasia severity in early stroke. The increased perfusion adjacent to the lesion may be crucial for early recovery in aphasia. Critically hypoperfused tissue within the central ischemic region may have a more rapid evolution to irreversible damage and a shorter therapeutic time window for tissue salvage than tissue bordering this zone, in which additional secondary and delayed cellular mechanisms may underlie progression to cell death. This model of penumbral evolution supports early reperfusion but indicates that the location of the penumbra and its distribution in gray and white matter compartments, may influence the choice of adjunct therapeutic strategies such as neuroprotection and the time window for their effectiveness. Subsequent language recovery and the long-term recovery in aphasia may be related to slow and gradual compensatory functions in the contralateral hemisphere, specifically in the homotopic frontal and thalamic areas.

Elizabeth Warburton et al in their PET study on the mechanisms of recovery from aphasia found that even limited salvage of peri-infarct tissue with acute stroke treatments will have an important impact on the rehabilitation of cognitive functions.

Reorganization of structure and function through the expression of neural plasticity plays a crucial role in recovery of language at least during the subacute phase of weeks to months after the occurrence of an injury.

Meinzer et al found Intensive language training enhances brain plasticity in chronic aphasia and the significance of perilesional areas in the rehabilitation of aphasia even years after the stroke.

7. SUMMARY AND CONCLUSION

Recovery pattern of thirty patients with aphasia were studied over a period of six months by measuring language performance at 4th week,8th week, 16th week and 24th week using tamil version of modified western aphasia battery in consultation with speech therapist and specialists.

- Maximum recovery was noted in patients with transcortical motor aphasia and conduction aphasia. Out of three patients with conduction aphasia, two transformed into anomia. Patients with transcortical motor aphasia became normal.

- Patients with broca's aphasia showed fair recovery. Two patients out of nine evolved into transcortical motor aphasia.

- Even though, global aphasia showed poor recovery, auditory word recognition was noted to improve. Three evolved into broca's aphasia.

- Among showing recovery groups, significant improvement was noted from 8th week of stroke.

- Initial severity and final outcome correlated significantly. Patients with initial high scores indicating mild involvement improved better.

- There was good correlation with the clinical –anatomical location of lesion and CT scan brain.

The neurolinguistic aspect of the speech recovery in stroke patients and psychosocial aspects of stroke patients are further continued as an ongoing study in our hospital and are still under follow up as a continuation of the present study.

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9. APPENDIX

PROTOCOL

STUDY OF RECOVERY PATTERN OF APHASIA IN STROKE PATIENTS

Name: Age: Sex:

Address:

Language:

Handedness:

Education:

Occupation: Risk Factors: Systemic diseases:

Present illness: Onset and course:

Signs Hemiplegia Recovery: Mild

Hemianopia Moderate

Sensory loss Severe

Investigations Date:

C.T.Scan

INSTITUTION: Coimbatore Medical College Hospital

Follow up:

I. SPONTANEOUS SPEECH

1. eP';fs; ,d;W vg;go ,Uf;fpwPh;fsh>
2. eP';fs; Kd;g[,';F te;jpUf;fpwPh;fsh>
3. c';fs; bgah; vd;d>
4. c';fs; tPl;L tpyhrk; vd;d>
5. c';fs; ntiy vd;d>
6. c';fSf;F vd;d f#;lk; cs;sJ>
7. ,e;jg; glj;ijg; ghh;j;J bjhpe;jijr; brhy;Y';fs; **:**

Information content: 10 Fluency, Grammatical Competence and Paraphasla-10

Max. Score : 20

Patient Score:

II. AUDITORY VERBAL COMPREHENSION

1. **Yes or No Questions : (20 Questions)**

Max. Score : 60

Patient's Score :

2. **Auditory Word Recognition**

Real Objects	**Drawn Objects**	**Forms**	**Letters**	**Numbers**
ngdh	tPL	rJuk; g	**10**	
fhR	ghl;oy;	Tl;;ly; k	**25**	
iff;fofhuk;	g{	tl;lk; m	**1438**	
kzpgh;!:	kdpjd;	el;rj;jpuk; X		**7**
lhh;riyl;	fofhuk;	Kf;nfhzk; f	**300**	
rhtp	fg;	mk;g[f;Fwp c		**6**

Colours	Furnitures	Body parts	Fingers	Right –Left parts
rpfg;g	rd;dy;	fhJ	fl;il tpuy;	tyJ-njhs;gl;il
gr;ir	ehw;fhyp	\f;F	nkhjpu tpuy;	,lJ fz;
ePyk;	nkir	fz;	Rz;L tpuy	tyJ kzpf;fl;L
k";rs;	fjt	jhil	Ms;fhl;otpuy;	,lJ fqf;fhy;
fUg;g[	Tiu	fGj;J	eLtpuy	tyJ fhJ
Cjh	!:?y]	khh;g	fhy;fl;il tpuy]	,lJ KH';if

Max. Score:60

Patient's Score:

3. Sequential Commands

1. ifia J}f;F **(2)**
2. fz;iz \L **(2)**
3. ehw;fhypiaf; fhz;gp **(2)**
4. $d;diyf; fhz;gpj;J tpl;L fjitf; fhz;gp **(4)**
5. ngdhita[k; g[j;jfj;ija[k; fhl;L **(4)**
6. ngdhthy; g[j;jfj;ijf; fhl;L **(8)**
7. g[j;jfj;jhy; ngdhitf; fhl;L **(8)**
8. ngdhthy; mst[nfhiyf; fhl;L **(8)**
9. g[j;jfj;jhy; mst[nfhiyf; fhl;L **(8)**
10. g[j;jfj;jpd; nky; ngdhit itj;J vd;dplk; bfhL **(14)**
11. ngdhtpd; gf;fj;jpy; mst[nfhiy itj;J g[j;jfj;ij mjd; nky; it. **(20)**

Max, Score : 80

Patient's Score:

III. REPETITION

1.	gh!;	**2**	
2.	\f;F	**2**	
3.	fz;	**2**	
4.	$d;dy;	**2**	
5.	thiHg;gHk;	**4**	
6.	iff;;fofhuk;	**4**	
7.	ehw;gj;jp Ie;J	**4**	
8.	bjhz;qw;W Ie;J rjtpjk;	**6**	
9.	,uz;L kzp Ik;gj;J Ie;J epkpl';fs;	**10**	
10.	kzp moj;Jf; bfhz;L ,Uf;fpwJ	**8**	
11.	flnyhuj;jpy; ge;J cUSJ g[uSJ	**10**	
12.	kpd;Dtbjy;yhk; bghd;dy;y	**8**	
13.	ahidf;bfhU fhyk; te;jhy; g{idf;bfhU fhyk; tUk;	**10**	
14.	ehd; filf;Fg; ngha; kpl;lha; th';fpndd;	**10**	
15.	nfhitapypUe;J nryk; fpnyhkPl;lh; bjhiytpy; cs;sJ	**160 100**	**20**

Max Score :100

Patients Score

IV. NAMING

a). Object Naming :

1. iff;fofhuk; **2**. ngdh **3.** g[j;jfk; **4.** fhR **5.** rhtp **6**. mst[nfhy; **7.** lhh;r; iyl; **8.** ,d;r; nlg; **9**.kzp gh;!: **10.** nrhg;g[lg;gh **11.** jPg;bgl;o **12.** ?j; gpu#; **13**. g{l;L **14.** rPg;g[**15.** !;g{d; **16**. ,';f; ghl;oy; **17**. nfrl; **18**. tpf;!: lg;gh **19.** gt[lh; od; **20.** gy;g[

Max Score :60

Patient's Score

b) Word Fluency: cdf;Fj; bjhpe;j kpUf';fs; bgaiuf; TW

Max Score :20

Patient's Score

c) Sentence Completion

1. ghypd; epwk; ……..

2. Fsj;jpy; ……

3 ngdhtpdhy;…….

4. bgh';fy; bfhz;lhLk; khjk; ……

5. fhw;Ws;s nghnj ……

Max Score :10

Patient's Score

d) RESPONSIVE SPEECH:

1. vjdhy; vGjyhk;>

2. ghypd; epwd; vd;d>

3. xU thuj;jpw;F vj;jid ehl;fs;>

4. g!:i! ahh; Xl;Lfpwhh;>

5. !;lhk;g[fs; v';F th';fyhk;>

Max Score :10

Patient's Score

SCORE

Language Parameters	Maximum Score	Patient's subscores	Total for AQ
1. Spontaneous Speech			
Information content	10		
Fluency	10		
Total	**20**		
II. Comprehension			
Yes-No questions	60		
Auditory Word	60		
recognition	80		
Sequential commands			
Total	**200**		
Divided by 20	10		
III. Repetition	**100**		
Total	**100**		
Divided by 10	10		
IV. Naming			
Object Naming	60		
Word fluency	20		
Sentence completion	10		
Responsive speech	10		
Total	**100**		
Divided by 10	10		

Aphasia Quotient: Add and multiply the total by 2.

SPONTANEOUS SPEECH AND TEST SCORES

Case No.	Type Aphasia	Fluency (Max. Score 10)				Information Content (Max. Score 10)			
		T1	T2	T3	T4	T1	T2	T3	T4
1.	Global	0	0	0	1	0	0	0	0
2.	Global	0	0	0	0	0	0	0	0
3.	Global	0	0	0	0	0	0	0	0
4.	Global	0	1	2	2	0	1	3	3
5.	Global	1	1	2	3	0	0	0	0
6.	Global	0	0	1	2	0	0	0	0
7.	Global	1	3	4	5	1	3	4	5
8.	Global	0	4	5	6	0	5	7	8
9.	Global	0	5	5	7	0	6	7	8
10.	Global	0	0	0	0	0	0	0	1
11.	Global	1	1	2	3	0	0	0	0
12.	Broca	4	4	5	9	3	4	4	4
13.	Broca	3	4	4	4	3	4	4	4
14.	Broca	3	4	4	4	1	2	3	4
15.	Broca	1	1	2	2	0	0	1	1
16.	Broca	4	4	5	9	5	6	7	9
17.	Broca	1	3	3	4	1	2	3	4
18.	Broca	1	1	2	2	0	0	1	1
19.	Broca	1	3	3	4	1	2	3	4
20.	Broca	1	3	3	4	1	2	3	4
21.	Conduction	9	9	10	10	10	10	10	10
22.	Conduction	5	6	9	10	8	10	10	10
23.	Conduction	5	6	9	10	8	10	10	10
24.	Wernicke	8	8	8	8	1	3	3	3
25.	Wernicke	7	8	8	8	0	1	1	3
26.	Wernicke	7	8	8	8	0	1	1	3
27.	Wernicke	7	7	7	7	0	0	0	1
28.	Wernicke	7	7	7	7	0	0	0	1
29.	TCM	2	4	6	9	3	5	8	9
30.	TCM	2	4	6	9	3	5	8	9

T1 – 4 Weeks, T2 – 8 Weeks, T3 – 16 Weeks, T4 – 24 Weeks

COMPREHENSION AND TEST SCORES

Case No.	Type Aphasia	Test Score (Max Score 10)			
		T1	T2	T3	T4
1.	Global	1.4	1.8	2.1	2.1
2.	Global	1.1	1.2	1.3	1.3
3.	Global	1.1	1.8	2.0	2.0
4.	Global	1.4	1.7	1.8	2.0
5.	Global	1.4	1.8	2.3	3.0
6.	Global	1.3	1.9	2.1	2.1
7.	Global	1.5	7.5	8.0	9.0
8.	Global	3.1	4.8	7.6	8.0
9.	Global	3.0	4.7	7.5	8.2
10.	Global	1.4	1.8	2.1	2.1
11.	Global	1.4	1.8	2.3	3.0
12.	Broca	1.72	10.0	10.0	10.0
13.	Broca	7.5	8.0	8.0	8.5
14.	Broca	7.5	8.0	8.0	8.5
15.	Broca	8.0	8.5	8.5	8.5
16.	Broca	9.0	10.0	10.0	10.0
17.	Broca	7.5	8.0	8.0	8.5
18.	Broca	8.0	8.5	8.5	8.0
19.	Broca	8.0	8.6	8.5	8.8
20.	Broca	7.5	7.8	8.0	8.5
21.	Conduction	9.0	10.0	10.0	10.0
22.	Conduction	10.0	10.0	10.0	10.0
23.	Conduction	10.0	10.0	10.0	10.0
24.	Wernicke	0.5	1.5	1.8	10.0
25.	Wernicke	1.4	1.6	2.0	2.0
26.	Wernicke	1.6	2.0	2.0	2.1
27.	Wernicke	1.8	2.0	2.0	2.2
28.	Wernicke	1.6	2.0	2.0	2.0
29.	TCM	9.6	9.8	10.0	10.0
30.	TCM	9.5	9.7	9.8	10.0

REPETITION AND TEST SCORES

Case No.	Type Aphasia	Test Score (Max Score 10)			
		T1	T2	T3	T4
1.	Global	0	0	0	0
2.	Global	0	0	0	0
3.	Global	0	0	0	0
4.	Global	0	4	0	6
5.	Global	0	0	0	0
6.	Global	0	0	0	0
7.	Global	0.6	0.3	4.6	6.0
8.	Global	3.6	4.2	5.4	6.0
9.	Global	3.6	4.0	5.0	6.0
10.	Global	0	0	0	0
11.	Global	0	0	0	0
12.	Broca	0.6	3.0	4.5	6.0
13.	Broca	0.6	6.6	7.0	8.2
14.	Broca	1.0	1.6	3.6	5.0
15.	Broca	0.8	1.0	1.0	2.0
16.	Broca	6.0	8.0	8.0	8.0
17.	Broca	1.5	1.6	1.6	1.7
18.	Broca	0.8	1.0	1.0	2.0
19.	Broca	1.0	1.6	3.6	5.0
20.	Broca	3.0	5.0	5.0	6.6
21.	Conduction	4.8	6.0	7.0	7.6
22.	Conduction	3.0	6.0	9.2	9.2
23.	Conduction	2.6	3.8	4.0	4.0
24.	Wernicke	0.8	0.8	0.8	1.0
25.	Wernicke	1.4	2.0	2.0	2.2
26.	Wernicke	2.4	3.0	3.0	3.0
27.	Wernicke	8.2	8.4	8.6	10
28.	Wernicke	8.2	8.4	8.6	10.0
29.	TCM	2.4	3.0	3.0	3.0
30.	TCM	3.0	6.0	9.2	9.2

NAMING AND TEST SCORES

Case No.	Type Aphasia	Test Score (Max Score 10)			
		T1	T2	T3	T4
1.	Global	0	0	0	0
2.	Global	0	0	0	0
3.	Global	0	0	0	0
4.	Global	0	0	0	0.5
5.	Global	0	0	0	0
6.	Global	0	0	0	0
7.	Global	0.5	1.8	3.6	5.0
8.	Global	0	1.4	1.8	2.0
9.	Global	0	1.4	1.8	2.0
10.	Global	0	0	0	0
11.	Global	0	0	0	0
12.	Broca	4.2	4.4	5.0	8.6
13.	Broca	4.2	4.4	5.0	8.6
14.	Broca	0.5	1.2	2.5	3.5
15.	Broca	0	0	0	0.5
16.	Broca	4.2	4.6	5.0	8.8
17.	Broca	1.5	1.2	2.5	3.6
18.	Broca	0	0	0	0.5
19.	Broca	0.5	1.2	2.5	3.6
20.	Broca	0.5	1.2	2.6	3.6
21.	Conduction	2.7	4.0	9.4	9.4
22.	Conduction	3.0	4.0	6.8	6.8
23.	Conduction	0.3	1.2	1.2	1.2
24.	Wernicke	0	0	0	0
25.	Wernicke	0	0	0.4	0.9
26.	Wernicke	0	1	1	1
27.	Wernicke	2.2	4.8	6.4	8.0
28.	Wernicke	2.2	4.8	6.6	8.0
29.	TCM	0	1	1	1
30.	TCM	3.0	4.0	6.8	6.8

CONSENT FORM

STUDY OF RECOVERY PATTERN OF APHASIA IN STROKE PATIENTS

Please tick as appropriate

☐ I am the patient

☐ I am the patient's relative

I understand the following

1. The study is conducted in Coimbatore Medical College and Hospital with my full consent which is voluntary and free of any objections.

2. My doctor has explained to me all the necessary details of the study to which I am consenting willfully.

3. I appreciate that practice of science and medicine in the current scenario and I also knew that nothing is guaranteed for me in this study and no funds have been received by me in this study.

4. I understand, that complete anonymity cannot be guaranteed.

5. The material will not be used for advertising and packaging.

6. The material will not be used out of contest for ex. a picture will not be used to illustrate an article that is unrelated to the subject of the photograph.

Having understood all these details, I give my consent as a voluntary willful act.

Signed:

Date:

MASTER CHART

Case No.	Name	Age	Sex	Education (Standard)	Risk Factors	Associated Defects	T1 4Weeks	T2 8 Weeks	T3 16 Weeks	T4 24 Weeks	Initial type of Aphasia	Evolution at the end of 6 months	CT Scan
1.	Ramu	38	M	4th	Smoker	Rt Hemiparesis	2.8	3.6	4.2	4.2	Global	Global	Large infarct – Lt frontal, parietal & temporal lobes cortical & sub cortical Broca's, Wernicke's, supra marginal gyrus & adjacent areas.
2.	Somu	58	M	3rd	Smoker, HT	-do-	2.2	2.4	4.2	2.6	Global	Global	Infarct
3.	Mogan	38	M	5th	Smoker	-do-	2.2	3.6	4.0	4.0	Global	Global	Infarct
4.	David	43	M	4th	Smoker, HT, DM	-do-	3.0	8.0	16.0	18.0	Global	Global	Infarct
5.	Ameer	35	M	5th	Smoker	-do-	4.8	5.6	8.6	10.0	Global	Global	Infarct
6.	Bala	35	M	5th	Smoker	-do-	2.6	3.8	6.2	9.8	Global	Global	Infarct
7.	Sundari	52	F	8th	HT, DM	-do-	8.8	36.4	48.0	62.0	Global	Broca	Hemorrhagiclesion
8.	Kamalam	65	F	Uneducated	HT	-do-	13.4	38.6	54.0	60.0	Global	Broca	Hemorrhagiclesion
9.	Muthu	54	M	4th	HT, Alcoholic	-do-	13.6	38.8	56.0	64.0	Global	Broca	Hemorrhagiclesion
10.	Karthick	37	M	Uneducated	Smoker	-do-	4.8	5.8	8.8	12.0	Global	Global	Infarct
11.	Annamalai	48	M	Uneducated	DM, HT, Smoker	-do-	4.8	5.6	8.6	12.0	Global	Global	Infarct
12.	Arasu	46	M	Uneducated	Smoker, HT	-do-	60.2	70.4	70.8	95.2	Broca	TCM	Infarct in Lt. frontal lobe cortical and subcortical Broca's area and adjacent areas.
13.	Suppan	62	M	4th	Smoker, Alcoholic	-do-	36.0	48.0	43.0	52.0	Broca	Broca	Infarct
14.	Raja	35	M	Uneducated	Smoker	-do-	26.0	33.2	42.2	50.0	Broca	Broca	Infarct
15.	Kumar	48	M	6th	HT, Smoker, Alcoholic	-do-	21.0	23.0	25.0	28.0	Broca	Broca	Hemorrhagiclesion
16.	Subramani	55	M	Uneducated	Smoker, Alcoholic	-do-	56.4	65.2	70.0	90.4	Broca	TCM	Infarct
17.	Kaliappan	50	M	6th	Smoker, Alcoholic	-do-	22.0	31.0	40.0	50.0	Broca	Broca	Infarct
18.	Karuppan	58	M	5th	HT, Smoker	-do-	21.0	23.0	25.0	28.0	Broca	Broca	Hemorrhagiclesion
19.	Kuppusamy	62	M	4th	DM, Smoker	-do-	22.0	31.0	42.0	52.2	Broca	Broca	Infarct
20.	Krishnan	50	M	3rd	Smoker, DM	-do-	22.0	33.0	46.0	54.2	Broca	Broca	Infarct
21.	Kamatchi	56	F	5th	DM	-do-	81.7	88.0	92.8	94.0	Conduction	Conduction	Infarct Lt. temporal, parietal

M – Male

F – Female

HT – Hyper Tension

DM – Diabetes Mellitus

TCM – Trans Cortical Motor